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Profiting from closure: the private finance initiative and the NHS

A covert, untested, and destabilising way of restructuring health care

Private investment is efficient when it maximises the returns on capital. Public investment is efficient when it maximises returns within the constraints of public policy goals, like meeting the population's healthcare needs. Given these different aspirations, what does partnership between the private and public sectors mean in practice? Specifically, what happens when private finance funds hospital redevelopment, as the private finance initiative attempts to do? In a BMA report published this week, Declan Gaffney and Allyson Pollock of St George's Hospital Medical School address this question in what is the most detailed independent study to date of the 14 private finance initiative schemes approved in June this year.[1] Reliance on private investment, the authors say, inflates the scale of capital schemes to levels which far exceed more prudent public proposals as bidders try to improve their rate of return. This cost escalation puts new demands on public revenue, which in turn leads to the search for new economies and new subsidies. The economies inspire bed reductions and unpiloted innovations in healthcare provision, while the subsidies entail transfers from other health sectors and raids on the very public funds that private finance was meant to replace. Returns on capital come to predominate over other policy considerations, and the health service ends up paying more for less.

The figures are striking. From relatively modest beginnings, the estimated costs of the 14 schemes rose on average by 72% as investors proposed bigger schemes involving larger loans and more equity. In Swindon the cost rose by 229%. Hospital closures and bed reductions of 7% to 44% helped meet the cost by releasing land for sale and allowing economies to be made in the new buildings.

But asset sales and conjectured "efficiency savings" have proved inadequate to bridge the affordability problem which cost escalation had created, and a series of new subsidies have been introduced. Some health authorities increased their annual commitment, taking money from other schemes and from sectors, such as community services, most likely to bear the burden of cost shunting out of the hospital system. Regional offices translated block grant capital into revenue payments, which meant subsidising privately financed projects out of the equipment and maintenance budgets of hospitals wthout privately financed schemes. In several cases equipment replacement was dropped from the deals even though equipment formed part of the estimated capital cost. And finally, the NHS Executive introduced a direct annual subsidy for the first 30 years of the private finance contract, a subsidy almost large enough in the case of Swindon to have paid for the original public scheme which the private finance initiative scheme had replaced (£42m compared with £48m).

The private finance initiative, says the report, has been bailed out and the cost borne by other parts of the health service. It has become not just a mechanism for reducing hospital services but also a costly burden. This state of affairs is unlikely to be exposed by the system of appraising the initiative, as that overlooks the costs which the scheme shifts out of the hospital sector on to others.

These are important findings. They suggest that the private finance initiative results in commercial returns unduly influencing the conduct of capital planning and the determination of asset size. In Edinburgh, for example, efficiency savings tied to the proposed new hospital imply patient throughput approaching 88 finished consultant episodes per bed per year compared with a national average for England that has levelled out at 54.[2] There is no precedent in Britain for such levels of activity. The planning base, staffing, and resource implications of the new model of care on which the hospital depends are unclear and the practical arrangements remain unpiloted. This is not healthcare planning as it is traditionally understood.

The Department of Health knows this, of course, so why is it prepared to accept the cost and the risk? The signs are that the private finance initiative offers a vehicle for another agenda that is gaining ground among NHS managers. Under this agenda, largescale capital investment provides an opportunity to redesign the hospital sector. What are the problems to which large (and costly) capital investment is supposed to be a solution? In Birmingham, where the health authority started consulting last month on its own private finance initiative plan, the problems are said to be constantly increasing referrals to relatively expensive hospitals.[3] Their solution involves reducing the size of the hospital sector by half and substituting cheaper alternatives. This means building a new health infrastructure—which is where the private finance initiative comes in.

What Birmingham's analysis omits to mention is the role of capital charges in the pressure felt by hospital budgets. Capital charges, which force hospitals to earn commercial returns, were introduced by the previous government to make transfer to private health provision that much easier. This rump of a privatisation policy continues to influence the NHS asset base by encouraging ward and hospital closure in much the same way as the old window tax encouraged bricking up windows (D Mayston, paper in preparation). To pay for the privately financed project in Birmingham, the expected rate of return on redeveloped hospitals has been increased from the current 6% to 13%.[3] So hospital costs are artificially inflated rather than hospitals simply being too expensive.

The problem for Birmingham and Edinburgh, and the other areas which are using capital spending to drive out labour from the hospital sector, is that no one is yet clear what sort of health service will result or whether it will save money. In the past the impact of capital charges was felt by chronically sick and elderly patients. The impact of the private finance initiative will be wider as early discharge and prevented admission have their effects across the board. How will these patients react when they are directed to cheaper alternatives and what sort of care can they expect to find there?

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Preventing RhD haemolytic disease of the newborn

Give anti-rhesus(D) immunoglobulin to all pregnant women who are Rh negative

See p 1504

In 1994 we saw the 25th anniversary year of the worldwide introduction of anti-rhesus (D) immunoglobulin prophylaxis, one of the most successful prophylactic programmes in medical history. In 1977, 110 cases of stillbirth or neonatal death due to RhD haemolytic disease were registered in Britain; by 1992, this figure seemed to have dropped to nine cases.[1] In this issue of the *BMJ*, however, Whitfield and colleagues show that this is a serious underestimation (p 1504).[2] Extrapolation of their Scottish figures suggests that there are 50 deaths a year due to RhD haemolytic disease in Britain. They conclude that the discrepancy is due to underreporting because deaths due to early abortion (before 24 weeks) and late neonatal death (second to fourth week of life) are not included in official figures.

The policy introduced in 1969 was to give anti-D immunoglobulin to RhD negative women only after the birth of a RhD positive infant or after a sensitising event in pregnancy, such as antepartum haemorrhage. The question now is whether the policy should be extended so that all pregnant women who are RhD negative receive an antenatal dose of anti-D immunoglobulin. More than half the deaths in Whitfield's study were due to sensitisation of the mother between the 28th and 40th week of her first pregnancy. Routine antenatal prophylaxis with anti-D immunoglobulin would largely have prevented sensitisation in the first pregnancy, and neonatal death after a subsequent pregnancy. Another paper in this issue reports that introducing a programme of antenatal prophylaxis in Derbyshire reduced the sensitisation rate from 1.12% of women at risk to 0.28%, a finding that is consistent with previous work.[3-5] This paper also shows that the shift from hospital to community based antenatal care did not reduce the programme's success. The lower incidence of sensitisation might also have been due to a heightened awareness among general practitioners of the need to give anti-D immunoglobulin to women with antepartum haemorrhage; the number of such events doubled during the period under study.

There are two reasons why RhD immunisation and sensitisation of pregnant women still occurs. Firstly, because not all women receive anti-D immunoglobulin when they should, and, secondly, because small, undetected leaks of fetal blood into the maternal circulation can occur during the third trimester of pregnancy. Both issues were extensively discussed in April during a consensus conference organised by the Royal College of Physicians of Edinburgh and the Royal College of Obstetricians and Gynaecologists. Contributors were worried that current guidelines for prophylaxis with anti-D immunoglobulin were not being followed.[6 7] Potentially sensitising events during pregnancy such as blunt abdominal trauma and antepartum haemorrhage require that the mother is protected by an injection of anti-D immunoglobulin, preferably after a Kleihauer test or equivalent to asses the size of the fetomaternal bleed. Kleihauer tests may be difficult to perform and standardise,[8] but health professionals should at least recognise sensitising events and consider giving anti-D immunoglobulin "blind" if testing is not available. Pregnant women who are RhD negative should also be educated about the condition, so that they recognise the need for treatment should a sensitising event occur.

The conference agreed that the second cause of RhD immunisation—fetomaternal bleeding during the last trimester—would be largely eliminated by giving anti-D immunoglobulin antenatally in one of two possible dose schedules: two doses of 500 IU, one at 28 weeks and the other at 34 weeks, or one dose of 1000 IU given between 28 and 30 weeks. Both options are

effective.[3 9] Antenatal programmes are cost effective if reserved for women in their first pregnancy, as they were in the Derbyshire study.[10 11]

Although the conference consensus panel considered that this restriction could not be justified on ethical or economic grounds, it seems that the tremendous global shortage of anti-D immunoglobulin may prevent extension of antenatal prophylaxis to all pregnant women at risk. The shortage of anti-D immunoglobulin from voluntary RhD negative donors is a major concern worldwide. The risk of transmitting viruses with the immunising RhD positive red cells cannot be completely eliminated and has caused the withdrawal of many voluntary donors.[12] Most countries now accept immunoglobulin preparations from commercial sources, prepared from paid donors' blood, and these seem to be safe. Once human monoclonal anti-D immunoglobulin is available, shortage will be a thing of the past; but, while it is being developed and tested, the need for immunised donors will continue well into the next century, and the debate about how best to use this scarce resource will continue.

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Helicobacter pylori and its interaction with risk factors for chronic disease

We are not quite ready to recommend green tea and saki to the exclusion of coffee

See p 1489

In this issue Brenner and colleagues document the effects of lifestyle on *Helicobacter pylori* infection in 447 patients in a German rural practice (p 1489).[1] They found that the *H pylori* infection rate appeared to be reduced by alcohol consumption (≥100 g per week), increased by coffee drinking (≥3 cups per day), and non-significantly increased by smoking. In the past, when idiopathic gastric hyperacidity was considered to be the chief cause of dyspeptic symptoms, smoking, coffee, and alcohol were often implicated as exacerbating the condition and advice given to eliminate these habits. Increasingly since 1984, however, when Warren and Marshall hypothesised that campylobacter-like organisms were the cause of peptic ulcer disease,[2] there has been a need to re-evaluate the role of these traditional risk factors. This need has been addressed by Brenner et al with some seemingly unexpected results.

There are reasons why alcohol, coffee, and smoking might have little effect on, or even increase, the hostility of the gastric environment to *H pylori*. The acid gastric pH prevents most organisms from thriving or even surviving in the stomach. *H pylori*, however, has an electropositive internal milieu; twice the number of basic amino acids, argenine and lysine, as *Haemophilus influenzae* and *Escherichia coli*; and powerful urease activity, with the ability to produce both ammonia and factors that inhibit parietal cell acid production.[3] All these attributes make survival of *H pylori* in the stomach less influenced by the reduction in pH which may accompany coffee, smoking, and alcohol consumption.[4-6]

Alcohol and smoking tend to increase the rate of gastric emptying.[4 7] However, *H pylori* has at least five different adhesins to attach itself to gastric epithelial cells. These, together with the fact that the surface lipopolysaccharide of *H pylori* is several orders of magnitude less immunoganic than other enteric bacteria, may allow it to adhere to the gastric mucosal surface and create a quasi "stagnant loop syndrome" despite adequate luminal flow. These functions have been highlighted by the recent complete sequencing of the *H pylori* gene.[3] One of the remaining questions is why alcohol is effective in the face of this powerful bacterial genetic machinery. Is it the time honoured antiseptic effect of alcohol, to which *H pylori* has developed no defence?

H pylori infection is the most common human chronic bacterial infection, affecting half the population and associated with duodenal and gastric ulceration, chronic active and atrophic gastritis, gastric carcinoma, and mucosa associated lymphoid tissue lymphoma.[8] As well as the risk of cancer, Brenner et al draw attention to the role of *H pylori* in cardiovascular disease in a fashion similar to *Chlamydia pneumoniae*.[9] They speculate that the beneficial effect of low alcohol consumption on coronary heart disease may relate to its ability to reduce *H pylori* infection. A corollary might be that smoking and coffee consumption contribute to the risk of coronary heart disease by increasing susceptibility to *H pylori* infection. Furthermore, since coffee raises serum cholesterol concentrations unless it

is filtered to remove the offending diterpenes,[10] will filtered coffee lacking deterpenes still promote *H pylori* infection? Should we screen patients at high risk of coronary heart disease for *H pylori*? If so, can we use the more generally available IgG test requiring simply a serum sample or should a ^{13}C urea breath test be the standard? Finally, will alcohol consumption be advised to help eliminate *H pylori*?

Brenner et al outline the possible positive effect of alcohol and the negative effect of coffee on the elimination of *H pylori*. *H pylori* is important because of its association with a broad range of diseases. However, in view of its high prevalence, other factors, including genes and environment, are likely to be pivotal in the pathogenesis of disease. The high prevalence of this bacteria also raises the question of routine screening and, if so, which test to use. In addition, should changes in lifestyle to reduce *H pylori* infection be encouraged—for example, green tea and saki rather than coffee in Japan? Certainly, these data are the closest we have had to support St Paul's injunction to "take a little wine for thy belly's sake." The emphasis, however, may be on "little"—that is, 1-2 drinks daily. Above this level, despite the continued lower risk of coronary heart disease, the risk of cancer, stroke, and all cause mortality rises.[11 12] Finally, recent publications from the health professionals study of 47 806 men found no increased risk of duodenal ulcer associated with coffee, smoking, or alcohol[13] but a negative association with dietary fibre and vitamin A.[14] We require more studies before we are confident in giving advice on this important and complex topic.

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Pressure to prescribe

Involves a complex interplay of factors

See p 1506

Two thirds of consultations with general practitioners end with the issuing of a prescription.[1] The decision to prescribe is influenced by many factors, to do with the doctor, the patient, the doctor-patient interaction, and the wider social context, including the effects of advertising and the financial incentives and disincentives for all parties.[2-6] Hardline advocates of rational drug use do not look kindly on variations in prescribing patterns that cannot be explained by purely clinical factors.[1] The prescriber who allows the "Friday night penicillin" phenomenon to sway his or her clinical judgment tends to do so surreptitiously and with a guilty conscience.

But such behaviour is the rule rather than the exception. Several studies have shown that the prescribing behaviour of doctors is heavily influenced by their perceptions of the social background, beliefs, attitudes, and expectations of the patient,[2] as well as the uncertainty of the diagnosis.[5 7]

Bradley identified several patient factors associated with doctors' discomfort when prescribing (or refusing to prescribe) drugs: extremes of age and of social class, non-white ethnic group (because of perceived differences in expectations), patients with a medical or paramedical background, and patients whose history the doctor either did not know or knew only too well (frequent attenders, "heartsink," and "fat file" patients).[5]

Although the powerful placebo effects of drugs prescribed in such situations are well documented,[8] so are the maladaptive behaviours that follow. Patients who receive a prescription for a self limiting condition are more likely to expect (and receive) one if the same symptoms recur.[9] The profession is regularly called on to acknowledge its vulnerability to the allegedly intense pressure to issue a prescription when none is needed,[5] yet hard data on the extent of this pressure remain sparse. Patients originating from the Asian subcontinent in particular have been accused of attending their general practitioners expecting prescriptions for

"trivial complaints" and "ill defined conditions,"[10] and patients of Pakistani or Indian origin are more likely to be given a prescription than those from white and West Indian ethnic groups.[7]

Two recent studies in the *BMJ*,[11 12] one of them published this week, address the gap between the prescriptions patients actually expect and what their doctors assume that they expect. An Australian study confirmed previous findings that about half of all patients have clear expectations for a prescription.[11] After controlling for presenting condition and patients' actual expectations, doctors' perceptions of these expectations also independently influenced the decision to prescribe. A British study by Britten and Ukoumunne (p 000) found that doctors' perceptions of patients' expectations for a prescription were significantly related to patients' hopes and educational level, and to the broad category of diagnosis, as well as to characteristics of the individual doctor.[12] The decision to prescribe was strongly related to the doctor's perception of the patient's expectations, and, overall, doctors classified 21% of their own prescriptions as "not strictly necessary."

Cockburn and Pitt speculate that failure to ascertain patients' expectations is a major reason why practitioners prescribe more drugs in total than patients expect.[11] But although these two studies suggest that a doctor's assessment of a patient's expectation is wrong in about a quarter[11] and a sixth[12] of cases, neither study provides direct evidence that this misconstruction leads to substantial overprescribing. Of the 255 patients in Cockburn and Pitt's study who expressed their expectations as anything other than "Don't know," 71% were correctly classified by their doctor as either expecting or not expecting a prescription.[11] A further 21% were incorrectly classified as not expecting a prescription; despite this, half of them received one. Only 8% (20) of the 255 patients did not expect a prescription when their doctor thought they did, and 16 of these received one.

In Britten and Ukoumunne's study, doctors admitted feeling pressure to prescribe in 66 out of 540 encounters (12%), and were more likely to prescribe for this group of patients.[12] But the number of excess prescriptions given to this group (that is, the number beyond what would have been expected if there had been no perceived pressure to prescribe) amounted to 16 in 540 encounters and 5% of all prescriptions issued.

These two most recent studies concur with previous findings that patients who expect a prescription are many times more likely to receive one than those who do not. This evidence is compatible with the stereotype of demanding and manipulative patients repeatedly forcing the hands of their reluctant doctors. But it is also compatible with the fact that patients may have more insight into their medical needs than their doctors give them credit for, and that both doctor and patient may legitimately take account of non-medical factors in deciding whether a particular drug is necessary at a particular point in time.

The act of issuing a prescription is the culmination of a complex chain of decisions.[2] It is open to biomedical, historical, psychosocial, and commercial influences, no aspect of which can be singled out as the "cause" of non-rational prescribing. The search should continue for methods to measure the interplay of these disparate factors on the decision to prescribe.

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Evidence based practice in mental health

New journal acknowledges an approach whose time has come

Why has it proved so difficult to narrow the gap between research and practice in psychiatry and mental health? The provision of mental health services is determined by many factors, including government policy, public demand, the behaviour of general practitioners and mental health professionals, and the financial pressures under which purchasers and providers of services work. These groups often have widely disparate views about the nature of mental disorder and the most appropriate services, and many forces exist to keep their views apart. Now is the time for a different approach based on the optimum application of the available evidence—heralded by the publication early next year of a new journal, *Evidence- Based Mental Health*. This approach will not provide easy answers and there will still be room for discussion about interpretation of even the very best evidence. Nevertheless, an approach that, firstly, acknowledges that mental health services should be fundamentally evidence based and, secondly, helps

define what constitutes the best available evidence should clarify decision making.

The task is formidable. The public view depression as being mainly caused by life events, may be reluctant to consult their general practitioner, and believe that counselling is more effective than antidepressant drugs, which they consider to be addictive.[1] Differences also exist between the views of all the disciplines working with people with mental health problems. General practitioners, psychiatrists, clinical psychologists, and mental health nurses are educated and trained in unidisciplinary structures and organisations. These professional organisations often sustain interdisciplinary rivalries. Even within the single discipline of psychiatry considerable differences exist between clinicians who subscribe to different (and often competing) schools of thought.

How much evidence is available on which to base mental health services? In fact, there is much evidence, which, although often difficult to find, is gradually being systematically reviewed by organisations such as the Cochrane Collaboration.[2 3] Psychiatry was one of the first medical specialties to use extensively the randomised controlled trial, and one of the founding principles of the profession of clinical psychology in the 1950s was that practice should be based on the results of experimental comparisons of treatment methods. Multicentre randomised controlled trials showed the effectiveness of antidepressant and antipsychotic drugs in the 1960s.[4 5] The recognition of international variations in diagnostic practice led to the development of explicit diagnostic criteria such as the *Diagnostic and Statistical Manual*, third edition, of the American Psychiatric Association.[6 7] Methodological innovations such as meta-analysis were first used in health care by psychologists in psychotherapy.[8]

Despite these undoubted advances, however, a considerable gap remains between research and practice. For example, important variations exist in the treatment of depression, in the use of electroconvulsive therapy, and in the use of stimulant medication for attention deficit hyperactivity disorder.[9-11] In mental health nursing the recent increase in the amount of published research has rarely been reflected by changes in practice.[12 13] In clinical psychology it has been asserted that "in clinical practice empirically supported methods are routinely ignored in favour of intuition and clinical experience."[14] Moreover, the public perception of mental health services has not kept up with advances in research and practice.[1 9] Others have argued that mental health policy has usually been influenced more by political values than evidence.[15]

As elsewhere, one essential ingredient required to make mental health services clinically effective is to ensure that clinicians know how to use evidence. There are many workshops aimed at helping mental health clinicians of all disciplines acquire the skills required for evidence based practice. Clinicians also need easy access to high quality evidence.[16] In addition to the problems of keeping up to date common to all clinicians,[17] mental health practitioners are often geographically isolated from information resources. Each discipline has its own journals which, even if read, may not contain the most important research. To improve access to the best evidence as it is published the *BMJ* is starting a new journal, *Evidence-Based Mental Health*, in collaboration with the Royal College of Psychiatrists, the British Psychological Society, and the Royal College of Nursing. *Evidence-Based Mental Health* will be a sister journal to *Evidence-Based Medicine* and *Evidence-Based Nursing*, and *ACP Journal Club*, using the same methods and the same editorial office in the health information research unit at McMaster University. The aim of the journal will be to provide all mental health clinicians with the very best information about mental health care in the form of "value added" abstracts. The first issue will be in published in February 1998 and a launch conference will be held in London on 16 February 1998.

Evidence based practice offers a way of making sure that clinical practice is based on the best available evidence. But we also need a culture change with better integration of patient values into the implementation of research and a need to go beyond professional rivalries and other barriers to provide the best available care for patients.

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